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Lack of Prognostic Value of Type D Personality for Mortality in a Large Sample of Heart Failure Patients

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Background: Type D personality has been proposed as a prognostic indicator for mortality in cardiovascular disease. Most research examining this construct originates from one research group, and it is critical that the predictive value of Type D personality for adverse outcomes is independently cross-validated. This study examined its prognostic value in heart failure, relative to B-type natriuretic peptide (BNP) and depressive symptoms. **Methods:** We studied 706 patients with complete BNP, depressive symptom, and Type D personality and mortality data from 958 patients with heart failure enrolled after hospitalization for a multisite study of a disease management program. Multivariable models were adjusted for BNP and depression. **Results:** At 18 months, there were 192 deaths (27.2%). No evidence was found for a prognostic value of Type D personality in the unadjusted model (hazard ratio [HR] = 0.893, 95% confidence interval [CI] = 0.582–1.370). In contrast, BNP was significantly predictive of mortality (HR = 1.588, 95% CI = 1.391–1.812), whereas depression was not (HR = 1.011, 95% CI = 0.998–1.024). Type D was also not predictive in covariate-adjusted models (HR = 0.779, 95% CI = 0.489–1.242). Similar results were obtained when analyzing Type D as the interaction between continuous *z* scores of its two components, negative affectivity and social inhibition ($p = .144$). **Conclusions:** In the largest study to date, Type D does not predict mortality. Future research should construe Type D as the interaction of continuous negative affectivity and social inhibition *z* scores, rather than as a typology, and consider analyses replacing negative affectivity with depression.
Key words: Type D personality, heart failure, survival, B-type natriuretic peptide, depression.

BNP = B-type natriuretic peptide; **HF** = heart failure; **CES-D** = Center for Epidemiologic Studies Depression; **NYHA** = New York Heart Association.

INTRODUCTION

Starting with an often-cited *Lancet* article (1), Type D personality, which has been defined as the tendency to experience negative emotions and to inhibit self-expression in social interaction, has been proposed as a prognostic indicator for mortality in cardiovascular disease independent of biologic factors, including disease severity. However, the original *Lancet* study excluded deaths in the first 5 years, with an observation period of 6 to 12 years. After exclusion of these 93 patients, only 21 deaths remained to be explained, too few events to justify the multivariate regression analyses, which were thus overfitted, with a high risk of spurious findings (2). A series of subsequent studies mostly had small samples, inconsistent scoring of the Type D measure, varying start and length of follow-up periods, and overfitted regression equations, with six (3), eight (4), twelve (5) and four (6) deaths, respectively, being explained. These studies tended to have fewer events being explained than the number of covariates considered for entering into multivariate analyses. A later study reported on 47 deaths in a mean observation period of 30 months among patients with heart failure (HF) and found a significant effect for Type D (odds ratio = 2.16, 95% confidence interval = 1.05–4.43, $p = .04$) that did not persist when confounds were controlled (7). More recently, Type D was not found to predict 123 deaths among 641 HF patients in bivariate or multivariate analyses (8). It is noteworthy that, thus far, all studies relating Type D personality to mortality were conducted by the same investigator group, with the exception of one small study in which there were null findings, but only 11 deaths to explain (9).

Proposals have nonetheless been made for routine screening of patients with cardiovascular disease for Type D personality (10) and the use of Type D for stratification purposes (11). The clinical utility of this variable remains to be independently established. We undertook an evaluation of Type D personality as a predictor of mortality among HF patients, taking advantage of a large-scale clinical trial with almost as many events (death) as all previous Type D mortality studies combined. Assessments of patients were available with a biomarker, B-type natriuretic peptide (BNP), which has emerged as a reliable indicator of the severity of HF (12,13), and with depressive symptoms, an established prognostic indicator for clinical outcomes in coronary heart disease and HF (14), was also available.

MATERIALS AND METHODS

Study Design

This report draws on data from the previously reported Coordinating study evaluating Outcomes of Advising and Counselling in Heart failure (COACH) trial in the Netherlands (15,16), a multicenter, randomized controlled trial with blinded end point evaluation designed to evaluate the effects of disease management, that is, advising and counseling of HF patients. The COACH trial revealed no significant treatment effects on mortality (16). Patients were assessed for Type D personality at baseline (during hospitalization) and followed for mortality for 18 months thereafter.

Study Population

Patients were recruited between October 2002 and February 2005 while hospitalized for symptomatic HF (New York Heart Association [NYHA] II–IV). Patients were required to be at least 18 years old and have evidence of structural underlying heart disease. Patients with impaired and preserved left

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ventricular ejection fraction were included. Major exclusion criteria were concurrent inclusion in another study or HF clinic, inability to complete questionnaires, invasive procedures or cardiac surgery performed within the last 6 months or planned within the next 3 months, ongoing evaluation for heart transplantation, and inability or unwillingness to give informed consent. Of the 958 patients enrolled in the trial, 706 had complete data for BNP, depressive symptoms, and Type D personality, and so, they were included in the present analyses.

Once informed consent was signed, baseline data collection started, and afterward, patients were randomized into one of three groups: basic support, intensive support, or control. The study complied with the Declaration of Helsinki, and the protocol was reviewed and approved by a central appointed ethics committee.

Data Collection

Data on mortality were collected from medical records. All reported deaths were reviewed by an independent clinical end point committee who defined the date and cause of death. The time of death from point of randomization was entered into Cox proportional hazards regression models.

Type D personality was assessed at baseline using the Type D scale (14-item version [DS14]), consisting of two seven-item subscales, that is, negative affectivity and social inhibition (17). As customary, patients were defined as having Type D personality when they scored 10 or higher on both subscales. The DS14 is generally construed to measure two temporally stable personality traits, as indicated by good test-retest reliability, and to be independent from changes in mood (17).

Depressive symptoms were assessed at baseline with the Center for Epidemiologic Studies Depression (CES-D) Scale (18,19), a 20-item well-validated measure that is commonly used with cardiac patients. The scores range from 0 to 60, and a validated cut point of 16 or higher is typically used to distinguish between low and high levels of depressive symptoms.

BNP measurement in this sample is described elsewhere (20). Basically, BNP plasma levels were determined using a Triage fluorescence immunoassay kit (Biosite Inc., San Diego, CA) within 4 hours of blood collection on the day of hospital discharge or 1 day before hospital discharge. For simplifying interpretation, BNP values were divided by 1000. Patients with available BNP levels did not differ in demographic or clinical characteristics, and the rates of Type D personality were not significantly different (p = .56) between patients with available BNP levels (n = 721, 13%) and patients who did not have a BNP measurement (n = 237, 12%).

Statistical Analysis

Descriptive sample statistics for baseline characteristics of the whole sample were calculated, as well as the prevalence of Type D personality and the relationship between Type D classification and key variables. Bivariate associations of Type D classification and mortality were calculated.

A Cox proportional hazards regression model was constructed for Type D personality. BNP and depressive symptoms were entered as the first block in an equation predicting mortality, with the entry of Type D in the second block testing the hypothesis that Type D classification had significant added prognostic value. Other potential control variables were considered but had to meet the requirement of not only being related to both Type D personality and mortality but also as potentially preceding or determining both Type D personality and mortality. We thus did not evaluate potential mediators of Type D on mortality as confounders (21,22).

Taxometric analyses (23) suggest that Type D is better represented as a dimensional construct rather than a categorical construct. Moreover, there is a long-standing consensus among psychometricians and personality theorists

TABLE 1. Baseline Characteristics in Relation to Type D Personality

	Total Sample (n = 706)	Type D (n = 95, 13%)	Non–Type D (n = 611, 87%)	p
Demographic variables				
Age, M (SD), y	70.7 (11.5)	69.3 (12.3)	70.9 (11.3)	.203
Female sex, %	38.2	42.1	37.6	.405
Clinical variables				
LVEF, M (SD), %	33.8 (14.3)	34.7 (15.2)	33.7 (14.2)	.533
History of AF, %	33.4	34.7	33.2	.771
NYHA (at discharge), %				
II	49.4	37.6	51.2	
III–IV	50.6	62.4	48.8	.015
Ischemic etiology, %	43.2	45.3	42.9	.663
≥1 comorbidity, %	78.6	76.8	78.9	.651
Prior HF admission, %	33.4	34.7	33.2	.771
BNP, M (SD)	0.674 (0.72)	0.659 (0.61)	0.676 (0.74)	.697
Medication at discharge, %				
ACE/ARB	84.0	80.0	84.6	.254
Diuretics	96.5	96.8	96.4	.828
β-Blockers	65.2	60.0	66.0	.257
Lipid-lowering drugs	37.7	35.8	38.0	.683
Antidepressants	6.1	13.7	4.9	.001
Negative affectivity, M (SD)	6.4 (6.0)	15.8 (4.3)	4.9 (4.7)	
Social inhibition, M (SD)	7.9 (7.1)	16.8 (5.0)	6.5 (6.3)	
Depression				
CES-D, M (SD)	15.4 (10.7)	25.9 (10.7)	13.8 (9.7)	<.001
≥16, %	39.9	76.8	34.2	<.001

M = mean; SD = standard deviation; LVEF = left ventricular ejection fraction; AF = atrial fibrillation; NYHA = New York Heart Association; HF = heart failure; BNP = B-type natriuretic peptide; ACE/ARB = angiotensin-converting enzyme/angiotensin receptor blocker; CES-D = Center for Epidemiologic Studies Depression.

that the practice of dichotomizing two continuous variables and constructing a typology for a resulting 2 × 2 matrix of high-low groups is variously unnecessary, highly problematic, and prone to spurious associations and therefore should be avoided (24–27). We therefore also analyzed the arguably more appropriate prediction of mortality from *z* scores for the components negative affectivity and social inhibition and their interaction term.

RESULTS

Baseline Characteristics

The present analyses included 706 patients who had BNP assessments and CES-D scores and who completed all 14 questions of the DS14. Analyses of differences between the 706 patients and the larger sample of 958 participants in the COACH trial from which they were drawn revealed only that patients included in the study were lower in prescription of antidepressants, (6.1% versus 9.9%), *t* test, $p < .04$. In total, 95 patients (13%) in the present sample were identified as having a Type D personality. Baseline characteristics of the study sample are provided in Table 1. Mean age of the study sample was 70.7 years, and 38.2% of them were women. At hospital discharge, 49.4% of the patients were classified as having NYHA functional Class II disease, and 50.6% were classified as having NYHA III or IV disease. A total of 43.2% had ischemic HF with a history of a myocardial infarction. Type D classification was not associated with baseline characteristics with the exception of NYHA classification and use of antidepressants, and a strong association with depressive symptoms, whether measured dichotomously or with CES-D continuous scores. Pearson correlations between continuous CES-D scores and the two continuous components of Type D, negative affectivity and social inhibition, were 0.62 ($n = 706$, $p < .001$) and 0.34 ($p < .001$), respectively.

Table 2 provides baseline characteristics for both survival states. Survival was related to age, all clinical variables including angiotensin-converting enzyme/angiotensin receptor blocker medication, with the exception of percent left ventricular ejection fraction and other medication. The only variable that was significantly related to both Type D and survival was NYHA. Because we did not construe this variable as a determinant of Type D, there was no need to control for this variable in the Cox proportional hazards regression model.

Relationship Between Type D and Mortality

All cause mortality rate for the study sample was 27.1% ($n = 192$). A Cox proportional hazards regression model relating Type D classification to mortality was not significant. Figure 1 depicts survival curves for Type D versus non–Type D. Although not significant at 18 months, the advantage of Type D personality for survival would have to be reversed substantially for a disadvantage of Type D personality to emerge at some point beyond our 18-month observation period.

TABLE 2. Baseline Characteristics in Relation to Survival

	Total Sample ($n = 706$)	Dead ($n = 192$, 27%)	Alive ($n = 514$, 73%)	*p*
Demographic variables				
Age, M (SD), y	70.7 (11.5)	74.4 (10.0)	69.3 (11.7)	<.001
Female sex, %	38.2	33.9	39.9	.142
Clinical variables				
LVEF, M (SD), %	33.8 (14.3)	33.3 (14.5)	34.0 (14.3)	.622
History of AF, %	33.4	45.8	28.8	<.001
NYHA (at discharge), %				
II	49.4	38.9	53.3	
III–IV	50.6	61.1	46.7	.001
Ischemic etiology, %	43.2	51.0	40.3	.010
≥1 comorbidity, %	78.6	87.0	75.5	.001
Prior HF admission, %	33.4	45.8	28.8	<.001
BNP, M (SD)	0.674 (0.72)	0.952 (0.91)	0.570 (0.60)	<.001
Medication at discharge, %				
ACE/ARB	84.0	76.6	86.8	.001
Diuretics	96.5	97.9	95.9	.200
β-Blockers	65.2	59.9	67.1	.073
Lipid-lowering drugs	37.7	37.5	37.7	.953
Antidepressants	6.1	6.8	5.8	.644
Negative affectivity, M (SD)	6.4 (6.0)	6.2 (5.7)	6.4 (6.0)	.772
Social inhibition, M (SD)	7.9 (7.1)	7.8 (6.8)	7.9 (7.2)	.952
Type D personality, %	13.5	12.5	13.8	.649
Depression, M (SD)	15.4 (10.7)	16.4 (10.3)	15.0 (10.8)	.114
CES-D ≥16, %	39.9	44.3	38.3	.151

M = mean; SD = standard deviation; LVEF = left ventricular ejection fraction; AF = atrial fibrillation; NYHA = New York Heart Association; HF = heart failure; BNP = B-type natriuretic peptide; ACE/ARB = angiotensin-converting enzyme/angiotensin receptor blocker; CES-D = Center for Epidemiologic Studies Depression.

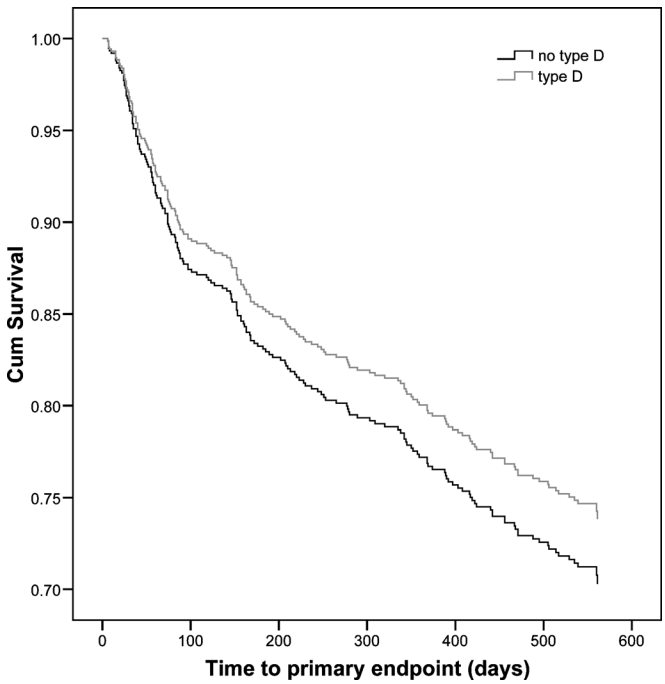

Figure 1. Survival for Type D.

As seen in Table 3, BNP levels and CES-D scores were entered as a first block in a Cox proportional hazards regression model, and the overall block proved a significant predictor of mortality, but this was because of the contribution of BNP levels, with the contribution of CES-D scores not significant (Model 1). In Model 2, the dichotomous variable Type D was added. This did not improve the prediction of mortality.

Table 4 again starts with a model with BNP levels and CES-D scores (Model 1). This time, in Model 2, the two components of Type D, negative affection and social inhibition (*z* scores), were entered together with their interaction. The interaction term was not significant in improving the prediction of mortality, again indicating that Type D did not contribute to the prediction of mortality when using continuous scores. To interpret the main effects of negative affection and social inhibition, the interaction term was removed, resulting in the final Model 3. BNP level seemed to be the best predictor of mortality. Depression also predicted mortality but to a limited degree.

DISCUSSION

No evidence was found for the prognostic value of Type D personality for all-cause mortality in HF patients, either in unadjusted Cox proportional hazards regression models or additive or independent of BNP in multivariate Cox proportional hazards regression models or as an effect modifier for BNP. These results held in analyses treating Type D as a dichotomous typology as well as in more appropriate analyses examining Type D for the interaction of negative affectivity and social inhibition. These results stand in contrast to what was obtained for the prognostic value of BNP. Suggestions that Type D personality be routinely assessed in HF patients or be used for stratification are premature, at least for the prediction of mortality in HF patients.

Type D classification was most strongly related to depressive symptoms and to treatment with antidepressants, a likely proxy for clinical depression. Although the developers of the Type D measure assert that Type D is independent of mood (17), there is a notable overlap in the content of measures assessing depressive symptoms and the two components of Type D, negative affectivity and social inhibition. Consistent findings that the components of Type D are related to depressive symptoms have led to suggestions that Type D and depressive symptoms are both facets of negative affectivity and that any prediction of clinically significant outcomes by Type D independent of depressive symptoms might, as has been suggested previously, be considered to be an artifact of creation of a Type D personality typology from variables that are essentially continuous (23,28). In the present sample, the correlation between CES-D and one component of Type D, negative affectivity, approached the maximum predicted from the respective reliabilities of the two scales.

Initial Cox proportional hazards regression model in our study were constructed consistent with all past studies testing the prognostic value of Type D for mortality, namely, with Type D treated as a typology with patients high in both negative affectivity and social inhibition being contrasted with the other three quadrants in a high-low, 2 × 2 cross tabulation of negative affectivity and social inhibition. Next, we obtained the same null results in our treatment of Type D for the interaction between continuous negative affectivity and social inhibition *z* scores. However, our second analytic strategy is more defensible and appropriate, given not only conclusions of taxometric analyses that indicate that Type D is best construed in continuous, dimensional terms rather than a typology but also a consensus that has emerged for more than 30 years in the psychometric and personality theory literature that typologies created from high-low, 2 × 2 crossings of continuous dimensional variables are inappropriate and prone to spurious findings (24–27). We find these arguments compelling and suggest that future Type D personality research adopt our analytic

TABLE 3. Cox Proportional Hazards Regression Model of Survival Status With Type D Dichotomized

							95% CI for Exp (*B*)	
	B	SE	Wald	df	*p*	HR	Lower	Upper
Model 1								
BNP	0.46	0.07	46.07	1	<.001	1.59	1.39	1.81
Depression	0.01	0.01	2.30	1	.130	1.01	0.997	1.023
Model 2								
BNP	0.46	0.07	45.12	1	<.001	1.58	1.38	1.81
Depression	0.01	0.01	3.38	1	.066	1.01	0.999	1.028
Type D	−0.25	0.24	1.10	1	.294	0.78	0.49	1.24

Continuous score for depression was used.
SE = standard error; df = degrees of freedom; HR = hazard ratio; CI = confidence interval; Exp = exponentiation of; BNP = B-type natriuretic peptide.

TABLE 4. Cox Proportional Hazards Regression Model of Survival Status With Type D as Interaction Between Negative Affectivity and Social Inhibition

							95% CI for Exp (*B*)	
	B	SE	Wald	df	*p*	HR	Lower	Upper
Model 1								
BNP	0.46	0.07	46.07	1	<.001	1.59	1.39	1.81
Depression	0.01	0.01	2.30	1	.130	1.01	0.997	1.023
Model 2								
BNP	0.46	0.07	43.27	1	<.001	1.58	1.38	1.80
Depression	0.02	0.01	5.01	1	.025	1.02	1.00	1.04
Negative affectivity (*z* score)	−0.10	0.10	1.12	1	.289	0.90	0.74	1.09
Social inhibition (*z* score)	−0.03	0.08	0.15	1	.695	0.97	0.84	1.13
Negative affectivity by social inhibition	−0.10	0.07	2.13	1	.144	0.90	0.79	1.04
Model 3								
BNP	0.46	0.07	43.38	1	<.001	1.58	1.38	1.81
Depression	0.02	0.01	4.92	1	.027	1.02	1.00	1.04
Negative affectivity (*z* score)	−0.14	0.10	2.01	1	.156	0.87	0.72	1.05
Social inhibition (*z* score)	−0.03	0.08	0.19	1	.659	0.97	0.83	1.12

Continuous score for depression was used.
SE = standard error; df = degrees of freedom; HR = hazard ratio; CI = confidence interval; Exp = exponentiation of; BNP = B-type natriuretic peptide.

strategy of focusing on the interaction of negative affectivity and social inhibition or explain why it is not being adopted.

In our present sample, depressive symptoms were not a significant predictor of mortality, although the association was in the expected direction. However, the present sample was limited to patients for whom both BNP and assessment of Type D personality were available ($n = 706$). These patients were drawn from a larger sample ($N = 938$) in which depressive symptoms were modestly associated with morality (HR = 1.169, $p = .02$) (20). The larger literature is mixed concerning the prediction of mortality in HF from depressive symptoms, particularly when mortality is examined separately, rather than simply treated as one aspect of a composite end point. A recent review of studies of the association of depressive symptoms and mortality in HF reported null findings for inpatient samples, but most studies of outpatients found an association (29). The present sample was recruited and assessed during an inpatient stay.

The strong association between one component of Type D, negative affectivity, and both depressive symptoms and use of antidepressants raises the possibility that, in addition to preserving the components of Type D as continuous variables, future research should examine whether depressive symptoms could be substituted for negative affectivity without any substantial loss in predictive power with respect to clinical variables. The association between depressive symptoms and cardiovascular outcomes is stronger and based on a more substantial literature than is the case for Type D. Routine screening for depression in cardiovascular patients has already been recommended by a number of professional organizations (30), even if the benefits of screening for cardiovascular outcomes are yet to be established (31). Furthermore, it is unlikely that calls for screening for Type D (11) will lead to the DS14 supplanting measures of depression. Non–mental health clinicians are notably averse to introducing and sustaining routine psychological screening (32) and report that even brief depression screening measures, such as the nine-item Patient Health Questionnaire, are too long (33,34). If, however, it could be shown that a brief, seven-item measure of social inhibition added substantially to the predictive ability of depressive symptoms, perhaps screening for social inhibition could be added to screening for depression.

Null findings from one large study might be contradicted by subsequent studies, but we note important limitations in the small studies that have been cited in support of a prognostic value for Type D personality with respect to mortality. Claims for the prognostic value of Type D may fit the pattern of other psychosocial variables purportedly predicting mortality, for instance, fighting spirit in the prediction of mortality of cancer patients (35). Specifically, initial claims are based on underpowered studies but could not be validated in subsequent large-scale studies with appropriate control of biomedical variables. Reasons for the rise, persistence, and ultimate fall of such hypotheses are undoubtedly varied. They likely include early positive results, capitalizing on chance or multivariate analyses where bivariate associations are not significant, and methodological limitations of the small studies, as well as publication bias (36,37). Supporting the hypothesis of a publication bias, we note that previous studies with positive findings have had 4 to 21 deaths to be explained, too few to justify the multivariate analyses that were used. Moreover, such a small number of deaths being explained in the individual studies could not be expected to generate a consistent pattern of positive findings unless an exceptionally large and unprecedented effect of personality on mortality was present.

This study had the advantage of an adequate sample size and being one of the first studies with regard to mortality conducted outside the original Type D investigator group. Some of the patients included in this study were from the same clinical settings providing patients to earlier studies, and the remaining

patients were drawn from the same larger, cultural and medical system context, the Netherlands. However, the prevalence of Type D personality (13%) was lower than in previous studies. It was nonetheless consistent across recruitment sites, including those involved in past studies of Type D. It is quite possible that recruitment to a disease management program such as COACH attracts a lower proportion of patients with Type D personality.

This study had the limitation of being a secondary analysis of a clinical trial not having been designed expressly to test the prognostic value of Type D. Its follow-up period was limited to 18 months, and we cannot exclude the possibility that effects of Type D personality on mortality are not apparent until later. However, emergence of a disadvantage of Type D for survival would require a substantial reversal of trends apparent up to 18 months. We are unaware of any plausible mechanism by which Type D should come into play after 18 months and affect in a clinically significant way the survival of the patients who survive until then.

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